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DWPI	fleurbaaij-g$.in.	2	L1

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NEWS 23	Jan 29	FSTA has been reloaded and moves to weekly updates
NEWS 24	Feb 01	DKILIT now produced by FIZ Karlsruhe and has a new update frequency

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=> s l1 and VEGF
L2 133 L1 AND VEGF

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L4 ANSWER 1 OF 10 MEDLINE DUPLICATE 1
ACCESSION NUMBER: 1998058949 MEDLINE
DOCUMENT NUMBER: 98058949 PubMed ID: 9395496
TITLE: Inhibition of vascular endothelial growth factor (**VEGF**)-induced endothelial cell proliferation by a peptide corresponding to the exon 7-encoded domain of VEGF165.
AUTHOR: **Soker S**; Gollamudi-Payne S; Fidder H; Charmahelli H; **Klagsbrun M**
CORPORATE SOURCE: Department of Surgery, Children's Hospital, Boston, Massachusetts 02115, USA.
CONTRACT NUMBER: CA37392 (NCI)
GM47397 (NIGMS)
SOURCE: JOURNAL OF BIOLOGICAL CHEMISTRY, **(1997 Dec 12)** 272 (50) 31582-8.
Journal code: HIV; 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States

IDS

Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199801
ENTRY DATE: Entered STN: 19980129
Last Updated on STN: 20000303
Entered Medline: 19980115

AB Vascular endothelial growth factor (**VEGF**) is a potent mitogen for endothelial cells (EC) in vitro and a major regulator of angiogenesis in vivo. VEGF121 and VEGF165 are the most abundant of the five known **VEGF** isoforms. The structural difference between these two is the presence in VEGF165 of 44 amino acids encoded by exon 7 lacking in VEGF121. It was previously shown that VEGF165 and VEGF121 both bind to KDR/Flk-1 and Flt-1 but that VEGF165 binds in addition to a novel receptor (Soker, S., Fidder, H., Neufeld, G., and Klagsbrun, M. (1996) J. Biol. Chem. 271, 5761-5767). The binding of VEGF165 to this VEGF165-specific receptor (VEGF165R) is mediated by the exon 7-encoded domain. To investigate the biological role of this domain further, a glutathione S-transferase fusion protein corresponding to the VEGF165 exon 7-encoded domain was prepared. The fusion protein inhibited binding of 125I-VEGF165 to VEGF165R on human umbilical vein-derived EC (HUVEC) and MDA-MB-231 tumor cells. The fusion protein also inhibited significantly 125I-VEGF165 binding to KDR/Flk-1 on HUVEC but not on porcine EC which express KDR/Flk-1 alone. VEGF165 had a 2-fold higher mitogenic activity for HUVEC than did VEGF121. The exon 7 fusion protein inhibited VEGF165-induced HUVEC proliferation by 60% to about the level stimulated by VEGF121. Unexpectedly, the fusion protein also inhibited HUVEC proliferation in response to VEGF121. Deletion analysis revealed that a core inhibitory domain exists within the C-terminal 23-amino acid portion of the exon 7-encoded domain and that a cysteine residue at position 22 in exon 7 is critical for inhibition. It was concluded that the exon 7-encoded domain of VEGF165 enhances its mitogenic activity for HUVEC by interacting with VEGF165R and modulating KDR/Flk-1-mediated mitogenicity indirectly and that exon 7-derived peptides may be useful **VEGF** antagonists in angiogenesis-associated diseases.

L4 ANSWER 2 OF 10 MEDLINE DUPLICATE 2
ACCESSION NUMBER: 96215040 MEDLINE
DOCUMENT NUMBER: 96215040 PubMed ID: 8621443
TITLE: Characterization of novel vascular endothelial growth factor (**VEGF**) receptors on tumor cells that bind VEGF165 via its exon 7-encoded domain.
AUTHOR: **Soker S**; Fidder H; Neufeld G; **Klagsbrun M**
CORPORATE SOURCE: Department of Surgery, Children's Hospital and Harvard Medical School, Boston, Massachusetts 02115, USA.
CONTRACT NUMBER: CA37392 (NCI)
GM47397 (NIGMS)
SOURCE: JOURNAL OF BIOLOGICAL CHEMISTRY, **(1996 Mar 8)** 271 (10) 5761-7.
Journal code: HIV; 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199606
ENTRY DATE: Entered STN: 19960627
Last Updated on STN: 20000303
Entered Medline: 19960620

AB Vascular endothelial growth factor (**VEGF**), a potent angiogenic

factor, uses two receptor tyrosine kinases, FLK/KDR and FLT, to mediate its activities. We have cross-linked 125I-VEGF165 to the cell surface of various tumor cell lines and of human umbilical vein endothelial cells. High molecular mass (220 and 240 kDa) and/or lower molecular mass (165 and 175 kDa) labeled complexes were detected depending on the cell type. The 220- and 240-kDa labeled complexes were shown to contain FLT and FLK/KDR receptors, respectively. On the other hand, the 165- and 175-kDa complexes did not seem to contain FLK/KDR or FLT but instead appeared to contain novel **VEGF** receptors with relatively low molecular masses of approximately 120 and 130 kDa. These receptors were further characterized in breast cancer MDA MB 231 cells (231), which did not form the high molecular mass complexes and which did not express detectable amounts of flk/kdr or flt mRNA. The 231 cells displayed one VEGF165 binding site, with a Kd of 2.8 x 10(-10) M and 0.95 1.1 x 10(5) binding sites per cell. By comparison, human umbilical vein endothelial cells had two binding sites, one with a Kd of 7.5 x 10(-12) M, presumably FLK/KDR, and the other with a Kd of 2 x 10(-10) M, a value similar to the **VEGF** binding sites on 231 cells. These lower affinity/molecular mass receptors on 231 cells cross-linked 125I-VEGF165 but not 125I-VEGF121. Accordingly, exon 7 of **VEGF**, which encodes the 44 amino acids present in VEGF165 that are absent in VEGF121, was fused to glutathione S-transferase (GST). The GST-**VEGF**-exon 7 fusion protein bound to heparin-Sepharose with a similar affinity as VEGF165 and inhibited the binding of 125I-VEGF165 to 231 cells. Cross-linking of 125I-GST-**VEGF**-exon 7 to 231 cells resulted in the formation of 150- and 160-kDa labeled complexes that presumably contained the 120- and 130-kDa lower affinity/molecular mass VEGF165 receptors. It was concluded that certain tumor-derived cell lines express novel surface-associated receptors that selectively bind VEGF165 via the exon 7-encoded domain, which is absent in VEGF121.

L4 ANSWER 3 OF 10 MEDLINE DUPLICATE 3
ACCESSION NUMBER: 96215007 MEDLINE
DOCUMENT NUMBER: 96215007 PubMed ID: 8621410
TITLE: Selective binding of VEGF121 to one of the three vascular endothelial growth factor receptors of vascular endothelial cells.
AUTHOR: Gitay-Goren H; Cohen T; Tessler S; **Soker S**; Gengrinovitch S; Rockwell P; **Klagsbrun M**; Levi B Z; Neufeld G
CORPORATE SOURCE: Department of Biology, Technion, Israel Institute of Technology, Haifa 32000, Israel.
SOURCE: JOURNAL OF BIOLOGICAL CHEMISTRY, **(1996 Mar 8)** 271 (10) 5519-23.
Journal code: HIV; 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199606
ENTRY DATE: Entered STN: 19960627
Last Updated on STN: 20000303
Entered Medline: 19960620
AB VEGF121 and VEGF165 are vascular endothelial growth factor splice variants

that promote the proliferation of endothelial cells and angiogenesis. VEGF165 contains the 44 additional amino acids encoded by exon 7 of the **VEGF** gene. These amino acids confer upon VEGF165 a heparin binding capability which VEGF121 lacks. 125I-VEGF165 bound to three vascular endothelial growth factor (**VEGF**) receptors on endothelial cells, while 125I-VEGF121 bound selectively only to the flk-1 **VEGF** receptor which corresponds to the larger of the three **VEGF** receptors. The binding of 125I-VEGF121 to flk-1 was not affected by the removal of cell surface heparan sulfates or by heparin. Both VEGF165 and VEGF121 inhibited the binding of 125I-VEGF121 to a soluble extracellular domain of the flk-1 **VEGF** receptor in the absence of heparin. However, heparin potentiated the inhibitory effect of VEGF165 by 2-3-fold.

These results contrast with previous observations which have indicated that the binding of 125I-VEGF165 to the flk-1 receptor is strongly dependent on heparin-like molecules. Further experiments showed that the receptor binding ability of VEGF165 is susceptible to oxidative damage caused by oxidants such as H2O2 or chloramine-T. VEGF121 was also damaged by oxidants but to a lesser extent. Heparin or cell surface heparan sulfates restored the flk-1 binding ability of damaged VEGF165 but not the receptor binding ability of damaged VEGF121. These observations suggest that alternative splicing can generate a diversity in growth factor signaling by determining receptor recognition patterns. They also indicate that the heparin binding ability of VEGF165 may enable the restoration of damaged VEGF165 function in processes such as inflammation or wound healing.

L4 ANSWER 4 OF 10 MEDLINE DUPLICATE 4
ACCESSION NUMBER: 97126566 MEDLINE
DOCUMENT NUMBER: 97126566 PubMed ID: 8971481
TITLE: Vascular endothelial growth factor and its receptors.
AUTHOR: **Klagsbrun M**; D'Amore P A
CORPORATE SOURCE: Department of Surgery, Children's Hospital, Harvard Medical School, Boston, MA 02115, USA.. klagsbrun@al.tch.harvard.edu
SOURCE: CYTOKINE AND GROWTH FACTOR REVIEWS, **(1996 Oct)** 7 (3) 259-70. Ref: 136
Journal code: CF7; 9612306. ISSN: 1359-6101.
PUB. COUNTRY: ENGLAND: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199703
ENTRY DATE: Entered STN: 19970407
Last Updated on STN: 20000303
Entered Medline: 19970321

AB Vascular endothelial growth factor (**VEGF**) is a prime regulator of endothelial cell proliferation, angiogenesis, vasculogenesis and vascular permeability. Its activity is mediated by the high affinity tyrosine kinase receptors, KDR/Fik-1 and Fit-1. In this article, recently discovered structural, molecular and biological properties of **VEGF** are described. Among the topics discussed are **VEGF** and **VEGF** receptor structure and bioactivity, the regulation of **VEGF** expression, the role of **VEGF** and its receptors in vascular development, and the involvement of **VEGF** and its

receptors in normal and pathological (ocular and tumor) angiogenesis.

L4 ANSWER 5 OF 10 MEDLINE DUPLICATE 5
ACCESSION NUMBER: 95393420 MEDLINE
DOCUMENT NUMBER: 95393420 PubMed ID: 7545086
TITLE: Peripheral blood T lymphocytes and lymphocytes infiltrating human cancers express vascular endothelial growth factor: a potential role for T cells in angiogenesis.
AUTHOR: Freeman M R; Schneck F X; Gagnon M L; Corless C; **Soker S**; Niknejad K; Peoples G E; **Klagsbrun M**
CORPORATE SOURCE: Urology Research Laboratory, Children's Hospital, Boston, Massachusetts 02115, USA.
CONTRACT NUMBER: CA37392 (NCI)
DK47556 (NIDDK)
SOURCE: CANCER RESEARCH, **(1995 Sep 15)** 55 (18) 4140-5.
Journal code: CNF; 2984705R. ISSN: 0008-5472.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199510
ENTRY DATE: Entered STN: 19951020
Last Updated on STN: 19960129
Entered Medline: 19951006

AB CD3+ peripheral blood T lymphocytes were evaluated for expression of vascular endothelial growth factor (**VEGF**), an endothelial cell mitogen and potent angiogenic factor. **VEGF** mRNA expression was confirmed in CD3+ cells and Jurkat cells, a human T-cell line, by reverse transcription-PCR and in CD4+ and CD8+ T cell subtypes by Northern blot hybridization. Steady-state levels of **VEGF** mRNA were inducible in CD3+ T cells by hypoxia, a known inducer of **VEGF** mRNA accumulation. Secreted **VEGF** was detected in CD4+ and CD8+ T cell- and Jurkat cell-conditioned medium, indicating that T lymphocytes are capable of exporting bioactive concentrations of **VEGF** into the extracellular space. Human prostate and bladder cancers (prostatic adenocarcinoma and transitional cell carcinomas) were evaluated for **VEGF** mRNA expression by in situ hybridization. Tumor-infiltrating lymphocytes (TIL), identifiable immunocytochemically as T cells, along with tumor cells in these cancers, expressed **VEGF** mRNA. TIL in bladder cancers could be labeled with a specific anti-**VEGF** mAb, indicating that TIL are likely to be able to secrete **VEGF** protein in situ at bioactive concentrations. The finding that peripheral T cells and TIL in human tumors synthesize a factor known to be a specific mediator of neovascularization suggests a role for T lymphocytes as cellular effectors of angiogenesis.

L4 ANSWER 6 OF 10 MEDLINE DUPLICATE 6
ACCESSION NUMBER: 94380049 MEDLINE
DOCUMENT NUMBER: 94380049 PubMed ID: 7522446
TITLE: Variations in the size and sulfation of heparin modulate the effect of heparin on the binding of VEGF165 to its receptors.
AUTHOR: **Soker S**; Goldstaub D; Svahn C M; Vlodavsky I; Levi B Z; Neufeld G
CORPORATE SOURCE: Department of Biology, Technion, Israel Institute of Technology, Haifa.
SOURCE: BIOCHEMICAL AND BIOPHYSICAL RESEARCH COMMUNICATIONS,

(1994 Sep 15) 203 (2) 1339-47.
Journal code: 9Y8; 0372516. ISSN: 0006-291X.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199410
ENTRY DATE: Entered STN: 19941031
Last Updated on STN: 20000303
Entered Medline: 19941018

AB The binding of the 165 amino-acid form of vascular endothelial growth factor (VEGF165) to the **VEGF** receptors of vascular endothelial cells was potentiated by heparin and heparan-sulfate, but not by other glycosaminoglycans. Heparin fragments of 16-18 sugar units inhibited the binding of 125I-VEGF165 to **VEGF** receptors, while fragments larger than 22 sugar units potentiated the binding. Over-sulfated heparin was a better potentiator of 125I-VEGF165 binding than native heparin. O-desulfated and N-desulfated heparins potentiated the binding to a lesser extent than native heparin. Heparin and N-desulfated heparin efficiently inhibited the binding of 125I-VEGF165 to alpha 2-macroglobulin, but surprisingly, O-desulfated heparin was an ineffective inhibitor. Since alpha 2-macroglobulin does not bind heparin, it follows that VEGF165 does not bind O-desulfated heparin efficiently. These results suggest that the mechanism by which heparin modulates the binding of VEGF165 to the **VEGF** receptors may require an interaction with cell surface heparin binding molecules.

L4 ANSWER 7 OF 10 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE 7
ACCESSION NUMBER: 1994:325769 BIOSIS
DOCUMENT NUMBER: PREV199497338769
TITLE: HB-EGF expression is decreased and **VEGF** is increased in the progression of normal to transformed prostate epithelia.
AUTHOR(S): Freeman, Michael R.; Uchida, Toshi; **Soker, Shay**; Blotnick, Sy; Raab, Gerhard; **Klagsbrun, Michael**
CORPORATE SOURCE: Child. Hosp./Harvard Med. Sch., Boston, MA 02115 USA
SOURCE: Journal of Cellular Biochemistry Supplement, (1994) Vol. 0,
No. 18D, pp. 221.
Meeting Info.: Keystone Symposium on Breast and Prostate Cancer II Lake Tahoe, California, USA March 14-20, 1994
ISSN: 0733-1959.
DOCUMENT TYPE: Conference
LANGUAGE: English

L4 ANSWER 8 OF 10 MEDLINE DUPLICATE 8
ACCESSION NUMBER: 93216730 MEDLINE
DOCUMENT NUMBER: 93216730 PubMed ID: 7681826
TITLE: Vascular endothelial growth factor is inactivated by binding to alpha 2-macroglobulin and the binding is inhibited by heparin.
AUTHOR: **Soker S**; Svahn C M; Neufeld G
CORPORATE SOURCE: Department of Biology Technion, Israel Institute of Technology, Technion City, Haifa.
SOURCE: JOURNAL OF BIOLOGICAL CHEMISTRY, **(1993 Apr 15)** 268 (11) 7685-91.
Journal code: HIV; 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States

Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199305
ENTRY DATE: Entered STN: 19930521
Last Updated on STN: 19960129
Entered Medline: 19930506

AB Vascular endothelial growth factor (**VEGF**) is a mitogen for cultured endothelial cells, and a potent angiogenic factor in vivo. Incubation of 125I-**VEGF** with human or bovine serum led to the formation of 125I-**VEGF** containing complexes that had a molecular mass greater than 300 kDa. These complexes were specifically immunoprecipitated with anti-human alpha 2-macroglobulin (alpha 2M) antibodies. Similar high molecular weight complexes were formed when 125I-**VEGF** was incubated with commercially available alpha 2M. The 125I-**VEGF**.alpha 2M complexes were resistant to boiling in the presence of SDS. The formation of 125I-**VEGF**.alpha 2M complexes was inhibited by iodoacetic acid, indicating that free sulfhydryl groups are required for complex assembly. Tryptic digestion of alpha 2M did not affect its **VEGF** binding ability. Tryptic digestion of 125I-**VEGF**.alpha 2M complexes on the other hand, resulted in the degradation of bound 125I-**VEGF**, indicating that alpha 2M does not protect bound 125I-**VEGF** from proteolytic digestion. The binding of 125I-**VEGF** to alpha 2M was partially inhibited by an excess of basic fibroblast growth factor. Other growth factors which bind to alpha 2M, such as platelet-derived growth factor and insulin, did not inhibit the binding of 125I-**VEGF**. The binding of **VEGF** to alpha 2M inhibited its receptor binding ability, indicating that alpha 2M may function as a **VEGF** removal and inactivation factor. Heparin and heparan sulfate, but not other glycosaminoglycans such as chondroitin sulfate, efficiently inhibited the binding of 125I-**VEGF** to alpha 2M. It is possible that heparin-like molecules released from extracellular matrixes could prevent the inactivation of **VEGF** by alpha 2M resulting in the potentiation of processes such as tumor angiogenesis.

L4 ANSWER 9 OF 10 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE 9
ACCESSION NUMBER: 1994:13671 BIOSIS
DOCUMENT NUMBER: PREV199497026671
TITLE: **VEGF**/VPF: The angiogenesis factor found.
AUTHOR(S): **Klagsbrun, Michael (1); Soker, Shay**
CORPORATE SOURCE: (1) Dep. Surg., Children's Hosp. Harvard Med. Sch., Boston,
MA 02115 USA
SOURCE: Current Biology, (1993) Vol. 3, No. 10, pp. 699-702.
ISSN: 0960-9822.
DOCUMENT TYPE: Article
LANGUAGE: English

L4 ANSWER 10 OF 10 MEDLINE DUPLICATE 10
ACCESSION NUMBER: 92210580 MEDLINE
DOCUMENT NUMBER: 92210580 PubMed ID: 1556117
TITLE: The binding of vascular endothelial growth factor to its receptors is dependent on cell surface-associated heparin-like molecules.
AUTHOR: Gitay-Goren H; **Soker S**; Vlodavsky I; Neufeld G
CORPORATE SOURCE: Department of Biology, Israel Institute of Technology, Technion City, Haifa.

SOURCE: JOURNAL OF BIOLOGICAL CHEMISTRY, **(1992 Mar 25)** 267 (9) 6093-8.
Journal code: HIV; 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199205
ENTRY DATE: Entered STN: 19920515
Last Updated on STN: 19970203
Entered Medline: 19920504

AB Vascular endothelial growth factor (**VEGF**) induces the proliferation of endothelial cells and is a potent angiogenic factor that binds to heparin. We have therefore studied the effect of heparin upon the interaction of **VEGF** with its receptors. Heparin, at concentrations ranging from 0.1 to 10 micrograms/ml, strongly potentiated the binding of 125I-**VEGF** to its receptors on endothelial cells. Scatchard analysis of 125I-**VEGF** binding indicates that 1 microgram/ml heparin induces an 8-fold increase in the apparent density of high affinity binding sites for **VEGF**, but does not significantly affect the dissociation constant of **VEGF**. Cross-linking experiments showed that heparin strongly potentiates the formation of the 170-, 195- and 225-kDa 125I-**VEGF**-receptor complexes on endothelial cells. At high 125I-**VEGF** concentrations (4 ng/ml), heparin preferentially enhanced the formation of the 170- and 195-kDa complexes. Preincubation of the cells with heparin, followed by extensive washes, produced a similar enhancement of subsequent 125I-**VEGF** binding. The binding of 125I-**VEGF** was completely inhibited following digestion of endothelial cells with heparinase and could be restored by the addition of exogenous heparin to the digested cells. The enhancing effect of heparin facilitated the detection of **VEGF** receptors on cell types that were not known previously to express such receptors. Our results suggest that cell surface-associated heparin-like molecules are required for the interaction of **VEGF** with its cell surface receptors.